

TRANSIENT SUDDEN BILATERAL VISUAL LOSS AFTER ORTHOTOPIC HEART TRANSPLANTATION: A CASE REPORT

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Abstract- Post cardiac surgery ophthalmic complications are uncommon, and sudden visual loss is one of the most important ophthalmic problems in these patients. We report a 54-year-old woman that suffered transient sudden bilateral visual loss seven days after orthotopic heart transplantation. In ophthalmologic examination positive findings were bilateral normal size pupils without direct and indirect pupillary light reflex and bilateral severe diffuse vasospasm of retinal arteries. Other ophthalmologic findings were normal. Problem lasts for 12 hours and resolved spontaneously. Funduscopic examination identified bilateral normal retina at this time and fluorescein angiography revealed normal retinal vasculature. This presentation suggests retinal vasospasm as an unusual benign cause of bilateral sudden visual loss after orthotopic heart transplantation.

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Key words: Sudden visual loss, heart transplantation, transient bilateral visual loss

INTRODUCTION

Post cardiac surgery ophthalmic complications are uncommon, and sudden visual loss is one of the most important ophthalmic problems in these patients (1-3). Postoperative sudden visual loss is divided into permanent and transient groups. There are several causes for transient visual loss such as amaurosis fugax, obstructions of the vertebral arteries, retinal vasospasm, increased intra-cranial pressure, retinal migraine and psychological problems. This case report suggests retinal vasospasm as a benign cause of transient bilateral sudden visual loss after orthotopic heart transplantation (OHT).

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CASE REPORT

A 54-year-old woman with three year history of exertional dyspnea and chest pain that deteriorated since five months ago was admitted in department of cardiovascular surgery.

Positive findings in her past medical history included diabetes mellitus for 10 years that was under control by diet, and hyperlipidemia that was treated with lovastatin. Histories of cerebrovascular accident or transient ischemic attack were negative. Also, personal or familial history for significant visual problems was negative. Psychological evaluation revealed mild to moderate depression. In physical examination mild respiratory distress, decreased respiratory sounds, mottled of the inferior one third of the posterior chest wall predominantly at the right side, grade 4 systolic murmur at the cardiac apex due to mitral regurgitation and early diastolic S3 sound was found.

Transient sudden bilateral visual loss after OHT

Laboratory data showed mild microcytic anemia with hematocrit value of 30 with normal WBC and platelet count. Electrolytes, renal and hepatic functions were all normal. Electrocardiogram showed sinus rhythm and left bundle branch block. Chest X-ray showed enlarged heart silhouette with pulmonary congestion and mild to moderate plural effusion predominantly at right side. Transthoracic echocardiographic examinations revealed dilated left and right ventricular chambers with severe left ventricular dysfunction (ejection fraction of 20%), and grade 3 mitral valve regurgitation. Coronary angiography demonstrated normal coronary arteries and cardiac catheterization revealed pulmonary arterial pressure of 35/12 mmHg. A diagnosis of dilated cardiomyopathy (end-stage heart failure, class 4 NYHA) was made and she underwent OHT. Her immunosuppressive regimen included cyclosporine, cellcept and prednisone, and her anticoagulating regimen contained enoxaparin with a dose of 40 mg/day.

Seven days after OHT she complained of sudden bilateral blindness. She denied experiencing headache, nausea, vomiting or ocular pain. In general physical examination, scalp tenderness, jaw claudication, cervical bruit, cardiac arrhythmia, sensory or motor neurological defects, and red eye were not found. Laboratory data at this time showed mild leukocytosis (WBC 13000, PMN 70%) and normal ESR (13 mm/hr). PT and PTT were 15 sec and 58 sec, respectively. To rule out the catastrophic causes of painless sudden blindness such as central retinal artery occlusion, central retinal vein occlusion and retinal detachment, ophthalmologic consultation requested.

Ophthalmologic examination showed bilateral no light perception visual acuity, normal size pupils without direct and indirect papillary light reflex and normal extraocular muscle motion (without pain or restriction). In slit lamp examination anterior segment was normal. Applanation tonometry revealed normal intraocular pressure (14 mHg). On funduscopy examination optic disc cup and foveal reflex were all normal, and despite a 10 year history of diabetes mellitus, no sign of proliferative or non proliferative diabetic retinopathy was found. Bilateral severely narrowed retinal arteries were predominant sign that was observed on funduscopy examination (Fig. 1).

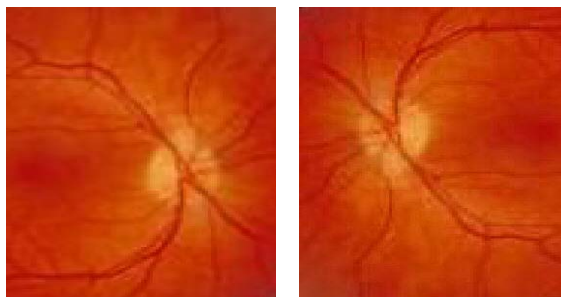


Fig. 1. Funduscopy examination showed severely vasoconstriction of bilateral retinal arteries.

Visual loss lasts for 12 hours, and then resolved spontaneously. Funduscopy examination showed normal retina at this time (Fig. 2). After return of vision to normal, fluorescein angiography revealed normal hemodynamic of retinal vasculature (Fig. 3).

DISCUSSION

Postoperative sudden visual loss can be a frightening complication, and all patients with a sudden loss of vision need emergency ophthalmic consultation (4, 5). Loss of vision refers to a severe blurring of the vision in one or both eyes often to the point that almost no detail can be made out. Because of the presence of underlying cardiovascular disease and the use of immunosuppressive regimen in OHT recipients, special attention to the altered visual acuity in these patients is required (6). Sudden visual loss is divided into transient and persistent categories and may be unilateral or bilateral. Etiologically, ischemia due to vascular insufficiency is the most common cause of sudden painless visual loss (7, 8, 9).

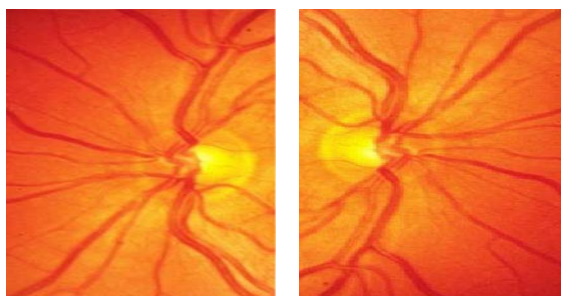


Fig. 2. After returns to the normal vision, ophthalmoscopic examination revealed normal vasculature of the fundus.

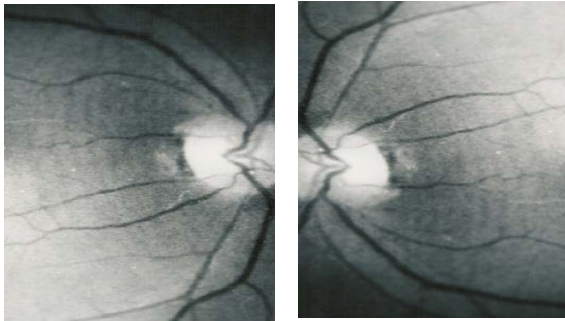


Fig. 3. Fluorescein angiograms, taken several minutes after infusion of contrast medium demonstrated normal vasculature by filling and passage of dye after returns to normal vision.

Persistent cause of sudden visual loss includes central retinal artery occlusion (8, 9, 16), central retinal vein occlusion (17), retinal detachment (18), vitreous hemorrhage (19), anterior ischemic optic neuropathy due to temporal arteritis (1, 20) and posterior ischemic optic neuropathies secondary to hypotension while under general anesthesia (2). Because the use of immunosuppressive regimen (cyclosporine, cellcept, and prednisone) in OHT recipients, some other permanent ophthalmic complications such as opportunistic infections, cyclosporine induced neurotoxicity and retinal toxicity (21-23), neovascular glaucoma, cataract and others must be expected (24).

There are several causes for transient visual loss such as amaurosis fugax, obstructions of the vertebral arteries, increased intra-cranial pressure, retinal migraine, retinal vasospasm and psychological problems. Amaurosis fugax refers to a temporary black-out of vision that usually affects one eye, is painless and is often described as a “shade coming down over the vision” of that eye. The black-out may last minutes, and then the vision returns (10, 11). People with obstructions of the vertebral arteries may notice temporary dimming of vision affecting both eyes, and possibly imbalance (12). Increase of intracranial pressure can cause momentary lapses of vision especially when moving, such as standing from a sitting position. Sometimes even eye movements are enough to induce a temporary loss of vision (13).

Retinal or ocular migraine is characterized by spasm of the artery supplies the retina. This spasm

can lead to a temporary black-out of vision on one side, and is fairly rare (15).

Vasospasm can have many different causes and can occur in a variety of diseases, including infectious, autoimmune, and ophthalmic diseases, as well as in otherwise healthy subjects.

Primary vasospastic syndrome and secondary vasospasm must be distinguished. The term “vasospastic syndrome” summarizes the symptoms of patients having such a diathesis as responding with spasm to stimuli like cold or emotional stress. Secondary vasospasm can occur in a number of autoimmune diseases, such as multiple sclerosis, lupus erythematosus, antiphospholipid syndrome, rheumatoid polyarthritis, giant cell arteritis, Behçet’s disease, Buerger’s disease and preeclampsia, and also in infectious diseases such as AIDS. Other potential causes of vasospasm include hemorrhage, major surgical procedures, homocystinemia, head injury, acute intermittent porphyria, sickle cell disease, anorexia nervosa, Susac syndrome, colitis ulcerosa, tumors, mitochondriopathies, Crohn’s disease, arteriosclerosis and drugs. The eye is frequently involved in the vasospastic syndrome, and ocular manifestations of vasospasm include alteration of conjunctival vessels, corneal edema, retinal arterial and venous occlusions, choroidal ischemia, amaurosis fugax, transient sudden visual loss, anterior ischemic optic neuropathy, and glaucoma. Since the clinical impact of vascular dysregulation has only been appreciated in the last few years, there has been little research in the according therapeutic field. The role of calcium channel blockers, magnesium, endothelin and glutamate antagonists, and gene therapy are discussed (11, 12).

Psychological cause of sudden visual loss must be suggested after ruling out other more important causes. Although our patient was moderately depressed, presence of significant abnormalities in ophthalmologic examination was suggestive of other causes of visual loss.

In summary we suggest that after ruling out other more important causes of postoperative blindness, retinal vasospasm should be considered as an unusual cause of bilateral sudden visual loss after orthotopic heart transplantation.

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